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Multiple Neuritis the Essential Element in Landry's Paralysis

An Illustrative Case

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MULTIPLE NEURITIS THE ESSENTIAL ELEMENT IN LANDRY'S PARALYSIS.[1]

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It seems probable that in the light of modern research the mysterious disease long known as Landry's paralysis will cease to exist as a distinct entity, though this term or its synonym, "acute ascending paralysis," may perhaps justly be retained as representing a group of cases, of extremely acute onset, and of characteristic distribution, of toxic origin, the essential lesion involving the nerves of the legs, trunk and arms, as well as the vagus and phrenic, implicating probably, at the onset at least, the spinal cord, and to a moderate extent the brain.

Classification of Landry's paralysis under spinal disease dates from a period when the spinal cord was the accredited seat of all paralysis, other than cerebral, and especially of all forms of paraplegia. The propriety of this classification is not established.

Multiple neuritis has steadily encroached upon the domain of the spinal cord in recent years, and in 1889 James Ross, in a series of masterly articles, demonstrated the practical clinical identity of Landry's paralysis with a well-recognized form of toxic neuritis, a doctrine which, though not accepted in its entirety, has on the whole gained ground, though few if any writers consider that multiple neuritis alone covers the

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entire pathology of the disorder. In the earlier description of Landry's paralysis we find complete relaxed paralysis of the legs, trunk and arms, of extremely rapid onset, with loss of reflexes (at a date when this symptom was studied), but with no disturbance of electrical reaction. There is little or no disturbance of sensibility, little or no disturbance of the sphincters, and no marked wasting. The prognosis is grave, the disease generally proving rapidly fatal through affection of the respiration.

This is certainly a clinical history, which, if taken literally, seems hard to reconcile with peripheral, though no harder than with central disease. Analysis of published cases show, however, that this history is incomplete, and later descriptions of the disease show considerable amplification. For example, out of 93 cases collected by Ross, we find that in 11 only is sensation definitely reported as unaffected, anesthesia with or without pain appearing in 22, paresthesia with or without pain in 25, tenderness alone in 3, hyperesthesia in 2, "fidgets" in 1, no mention of sensation in 15.

The proportion of cases in which sensation is unimpaired is, therefore, no greater than is reconcilable with peripheral disease, and the sensory disturbances in the larger proportion are quite characteristic of neuritis.

With regard to the electrical reactions, in 32 only of the cases quoted by Ross were the reactions tried, and in 12 of these the reaction of degeneration, complete or partial, was found. A careful analysis by this author of the cases remaining shows that in most instances the faradic current alone was used, "retained faradic irritability being quite compatible with that inversion of normal formula of galvanic reaction which constitutes the reaction of degeneration in its partial

form." It will be noted in our case that faradic contractility was normal in certain muscles (for example, right deltoid), in which the galvanic test showed inversion of normal formulæ (An Cl greater than Ka Cl).

In many of the cases the fatal onset occurs too early for reaction of degeneration to become established, a fact emphasized by that of Bernhardt, in which the early reactions were normal, but degeneration reaction appeared late in the progress of the disease.

It seems, therefore, that the two elements most difficult to reconcile with peripheral neuritis, that is, lack of sensory disturbances and tenderness, and of electrical impairment, are by no means essential elements of the disease, no more cases offering these peculiarities than are compatible with neuritis, in which it is known that sensory disorders may sometimes fail, and electrical reactions be found normal, at least at certain stages.

Now let us see how many of these cases conform to the classical type in respect to *both* loss of sensation and the electrical reaction. Out of the 11 cases without loss of sensation, in five no mention was made of the electrical reactions, in one faradic contractility was lost, in one galvanic reaction was altered, in one both affected, in three only the electrical reaction was mentioned as normal. In the 15 in which no mention of sensation was made, electricity was not used in 13, the reactions were found normal in one, and lost in one.

This leaves us four cases only out of 93 in which sensation was unaffected *and* electrical reactions were not impaired — a small number on which to base a classification. I have analyzed with reference to these points the 29 cases to which I had access out of the 48 published since the summary of Ross, with the follow-

ing result: sensation unaffected, five only; anesthesia, with or without pain, nine; paresthesia, eight; tenderness alone, one; pain alone, five; no statement regarding sensation, one.

Of the five cases without affection of sensation, in one there was no loss of electrical reaction (this expression probably meaning faradism), in the other four no note was taken of the reactions, adding therefore only one record to the list of cases following the generally accepted description of the disease, the electrical record even in that one case being incomplete and unsatisfactory.

With regard to the question of muscular wasting, it seems on looking over the recorded cases that wasting of muscles is apt to occur in the convalescent cases, not in the early fatal. But a glance at the period of fatality in those cases easily explains this anomaly — certainly neither muscular wasting nor degeneration reaction could be expected to appear in three days, the duration of one case in which their absence was noted, and even periods of six and nine days, as found in two others, are by no means enough to establish these points.

With regard to the question of fatality, analysis of 121 cases (including the 93 of Ross, and the 28 of the 29, of which I was able to find the termination, out of those reported since Ross's article) shows 77 fatal cases against 44 protracted recoveries, that is, 36 per cent. gradually recovered while 64 per cent. died. This is certainly a large percentage of deaths, but does not justify the grave prognosis ordinarily given.

It may be maintained that the fatal cases alone were worthy of being classed under Landry's paralysis, but the careful perusal of the records shows a similarity among the cases too striking to allow this claim. In all, the rapid onset, the complete involvement of the

trunk, the embarrassment of respiration, the total relaxed paralysis with loss of reflexes (where tested), and absence of marked sphincter weakness, were sufficiently conspicuous to justify forming a group, whether as a distinct class or as a subdivision of a class already established.

In respect to the etiology, it is a significant fact that in most of the cases in which a cause was assigned, that is, in about half, the cause was one generally recognized as giving rise to multiple neuritis; for example, alcohol, septicemic processes, syphilis and acute infectious diseases.

When we come to consider the pathological findings, the majority of autopsies may be disregarded as not sufficiently complete and accurate from a microscopical and bacteriological point of view to be conclusive. Certain it is, however, that various careful post-mortem examinations have revealed sufficiently marked changes to give a clue to the process; notably the following:

Centanni found marked interstitial neuritis and micro-organisms in the cord. Oettinger and Marinesco found nothing in the peripheral nerves, but in the gray matter of the spinal cord found infiltration of vessel walls with leucocytes, the latter filled with microbes (diplococcus), also with megarocytes with vacuoles and degeneration due to streptococcus. Eisenlohn found degeneration of peripheral nerves, also acute myelitic processes, and micro-organisms in the cord, as well as in the peripheral nerves. Pitres and Vaillard found marked degeneration of peripheral nerves, nothing in the cord. Baumgarten and Curschman found respectively, bacilli of malignant pustule and of typhoid in the cord. Hun, who opposes the view of neuritis, and regards the disease a clinical entity, found only a few degenerated fibres of the anterior roots of the cauda equina, and cerebral and spinal meningitis of recent origin.

In view of the frequent implication of the cord, as demonstrated by autopsy, it may be pertinently asked, why classify the disease under neuritis? But, conversely, it may be asked why, in view of the frequent implication of the peripheral nerves, should it be classed under spinal diseases? In other toxic disorders, notably lead-poisoning and diphtheritic paralysis, in which peripheral neuritis is considered the essential process, the cord is sometimes also invaded, and cases of so-called Landry's paralysis, though more acute, correspond much more nearly to this type of disorder than to disease of the cord. This is certainly true of the convalescent cases, which offer opportunity for careful study, and even in the cases rapidly succumbing to the overwhelming effects of the poison, signs are present that the nerves are also affected (pain and tenderness being frequently present) though the cord and brain are also attacked by the products of infection. The restlessness and hysterical tendency often noted at the onset certainly point to temporary involvement of the latter organ.

The cranial nerves are occasionally affected, though the cerebral symptoms are generally limited, if present, to those mentioned. In our case the restlessness was extreme, and temporary diplopia showed slight implication of the cranial nerves, other than phrenic and pneumogastric.

The foregoing considerations would seem to justify material modification of the disease. Perhaps it might fairly be described as follows:

Landry's paralysis is an acute toxic disease, characterized by rapid loss of power in the lower extremities, trunk, and to a less degree in the upper extremities, affecting also the vagus and phrenic, sometimes other cranial nerves. The affected muscles are lax. Pain, paresthesia, anesthesia, and tenderness are generally

present in varying degrees, though in some cases sensory disturbances are wanting. Death follows in more than half (64 per cent.) of the cases. Recovery when present is very slow. The reflexes, deep and superficial, are lost at an early stage; wasting and degeneration reaction appear if the patient survives. The process is a toxic affection of the peripheral nerves (neuritis), cord and brain, the former being the essential and persistent lesion.

These introductory observations will enable us to appreciate, on the one hand, how closely our case conforms in its essential characteristics to the type of so-called Landry's paralysis, and, on the other, how appropriately it falls under the head of the now well-recognized toxic multiple neuritis.

The discussion of its etiology I leave to those more learned in bacteriology than myself; but in view of a possible connection with the surgical experiences through which the patient has passed, I have invited Drs. Cabot and Harrington to be present, as well as Dr. Gannett, under whose care he came during the paralytic stage, and to whom I am greatly indebted for asking me to see the case in consultation, and for allowing me the opportunity to study and report it. I wish also to express my appreciation of the careful hospital reports for which the history is mainly taken, and specially those of Dr. Joslin, who followed the case with great care and completeness during the period which was of special interest from a neurological point of view.

The patient is a man about thirty years of age, of Irish extraction, a letter-carrier. Admitted into Dr. Cabot's service February 15, 1895.

Family History. — Father had kidney and asthmatic trouble; is said to have died of *la grippe*. Mother

suffered from nervous prostration, and is said to have died of heart failure. Two brothers and two sisters died in infancy. One brother is in good health but nervous; two sisters are living, not very strong.

Habits. — No tobacco. Gonorrhea at seventeen; again at twenty-four. No history of other venereal trouble.

Patient's History. — Has had scarlet fever, measles, varioloid, and whooping-cough; *la grippe* five or six years ago. As far back as he can remember, has had to get up at night to pass water; no incontinence. After the first attack of gonorrhea had the usual burning and frequency of micturition. Discharge lasted nine months. Was then perfectly well till second attack of gonorrhea. This attack was followed by the same symptoms, and lasted for about the same time. When in health has had to pass water every two hours. Beginning September, 1894, micturition was very frequent (from two minutes to three hours). He stopped work. Had chills and fever at night, and profuse sweating. There was flatulence at night. Slept fairly well. Appetite was poor; lost weight. On coming under Dr. Cabot's care had lost ten pounds since January 1, 1894. There was pain in the left renal region and in the left groin, and the kidney could be felt as a distinct tumor which was not tender to pressure. Jolting made micturition more frequent and increased the pain. There was a smarting sensation through the rectum and bladder after passing water. Has not been a heavy drinker, though a more or less steady one. Repeated examination of the urine failed to show tubercle bacilli.

Examination under ether by Dr. Cabot showed no stone on sounding.

On March 14th there was, on the whole, less pain than at entrance. The urine was of good quantity,

and contained less sediment. There was still much pus, small round cells and blood globules; albumin, a trace. Repeated examination showed no tubercle bacilli. He was transferred to Waverley, relieved.

He was re-admitted under Dr. Harrington, June 3, 1895, with the following history: Since discharged, symptoms have remained much the same, but has lost gradually a little in general health. Still abundance of pus in urine; pain in left side. Kidney can be felt on the left, enlarged and somewhat tender. Patient is very thin and anemic, though strong enough to walk about. Put to bed and watched. Alcohol with dinner. Strychnia, one-sixtieth, t. i. d. No tubercle bacilli in the urine. Dr. Harrington operated June 11th.

Operation. — Ether administered. An incision, four inches long, was made in the left flank, one inch above, and parallel with, the crest of the ilium. The incision was carried into the peri-nephritic fat, and the kidney isolated with the fingers. The surface of the kidney was exposed by dissection from the peri-nephritic fat over a space of several inches. Puncture with the aspirating-needle directly into the kidney substance revealed pus. An incision, two inches long, was made into the kidney substance, and was followed by a gush of pus. Washed out with boiled water. The finger introduced into the kidney felt calculus. This was withdrawn between the curette and the finger. Cavity douched with boiled water. Further exploration with the finger into the kidney showed tissue very friable and infiltrated with foul pus. Another large pocket of pus was found containing a calculus, and douched as before. In this way another pocket was opened, and five calculi in all, varying in size from a pea to a small marble, were removed. Several ounces of foul pus were removed and the cavity cleaned out as thoroughly as possible. At the end of the operation the

finger enters three cavities, the middle being the deepest and having a smooth lining supposed to be the dilated pelvis of the kidney. Drainage-tube surrounded with gauze inserted to the bottom of this cavity; the other two cavities packed with iodoform gauze. Dry dressing. Considerable shock after operation, but patient responded to stimulation with brandy (enema) and subcutaneous injection of strychnia.

Second day, patient very weak and all motions extremely painful. Much discharge, and dressing soaked with urine. Tube syringed with boracic acid, 1 to 40. Half an ounce of brandy every four hours.

Fourth day, still weak and motion painful. Pulse small and quiet. Strychnia, one-fortieth of a grain every three hours.

Seventh day, patient very comfortable. Pulse good. Passed 64 ounces of clear, pale urine. Dressing the same, but tube has been removed.

Ninth day, rapid general improvement. Has no pain, and moves freely. Is gaining flesh.

Twenty-fifth day, improvement seen every day in flesh and strength. Has been up and about for some time. A sinus three or four inches deep extends inwards and backwards. There is a line of granulations. Considerable discharge of pus, though free from pain. Patient discharged much relieved.

Re-admitted under Dr. Gannett's service August 28, 1895.

Last Friday night pain appeared in right lumbar region, and has been constant, though varying in intensity to time of entrance. Sunday night there was pain on both sides in the lumbar region. This extends through thighs to knees, also through to legs and buttocks. Pain in legs came Monday night, lasted twenty four hours. Yesterday morning lost control of both legs within a few hours. Can move toes only.

Sensation not affected. Vomited considerably Monday, but not since Tuesday morning. Diarrhea Monday, none since. No incontinence of feces, but has scalding movements. Urine passed in spurts. Catheterized yesterday afternoon by Dr. Harrington, and has not passed urine since. No cough, no palpitation. Yesterday and to-day has had chills and sweating lasting for a few minutes. Has had severe headache for three days. Has noticed no change in eyesight. Some twitching of legs, with an occasional cramp in calf of both legs. A sinus in left back at seat of wound has discharged since he left the hospital. Leucocytes 16,000 (3.25 P. M.). Urine: color normal, acid, 1020, slight uric-acid zone; albumin, one-eighth; sugar, none; urea, 2.08 per cent. Many hyaline and colorless fine granular casts, chiefly of small diameter. Some with a few renal or blood corpuscles adherent, occasionally blood corpuscles, normal or abnormal, free and on casts.

Physical Examination. — Man of small size, well developed, fairly well nourished. Pupils alike, and contract. Expression anxious and nervous. Lungs negative. Heart apex at fourth space, within nipple line. No murmurs. Abdomen: Below right costal border, two inches from median line, is a rounded body, which protrudes very slightly on inspiration, probably right kidney compensatorily enlarged. On left flank is a scar representing the seat of operation, with sinus from which a moderate amount of pus is discharged. Sensation perfect. All reflexes absent. Legs flaccid, not atrophied. In the afternoon dull headache with nausea and vomiting every half hour. Dr. Gannett considered patient uremic.

Pilocarpine, one-eighth of a grain, subcutaneously at 9 P. M.; blankets. Perspires freely. Nausea persisted till 11 P. M. Then slept three to four hours.

Third day (from onset of paralysis). No headache, no dulness. Sinus discharging freely. Potas. citrat., 60 grains every four hours. High oil injection, followed by enema of suds and glycerine.

Fourth day, passed 25 ounces of urine. No longer necessary to catheterize. Urine: color normal; reaction neutral; specific gravity, 1028; urea, 3.28 per cent.; albumin, one-eighth; sugar, none; sediment slight. Speech hitching. Diplopia. Partial paralysis of left arm, with complete paralysis of both legs, save for slight motion of toes of right foot. Cannot turn in bed. Constantly asking to have position changed on account of pain in legs. Not able with both hands to draw up foot by means of a bandage fastened on toes.

Fifth day, ophthalmoscopic examination by Dr. Amadon. Fundus normal. Reported very restless at night. Trional, 20 grains, in hot milk. Slept one and one-half hours.

Sixth day, restlessness continues.

Seventh day, so very restless that a special night-nurse was ordered, who reported: "Very nervous and restless from 8 to 1.45, urging to have position changed every two to ten minutes. Pain in thighs all night. From 1.45 A. M. to 5.30 called every twenty to thirty minutes to have position changed. Remainder of the time very nervous and depressed. Slept in all one hour and thirty-five minutes." Similar nights for about one week. No renal symptoms from this time. Sulphonal and trional, each ten grains, every night for four nights.

Fifteenth day, involuntary dejection this morning.

Eighteenth day, still involuntary dejection. Patient cannot cough.

At this time the complete physical condition (nervous) is as follows: The limbs are perfectly flaccid

and absolutely powerless. Both legs are quite cool, except slight plantar flexion of toes. There is tenderness of the sciatic and peroneal nerves, and in the muscles of the calf and thigh. The lightest touch is everywhere felt, though patient states that the electricity is not so plainly felt over the legs, as, for example, on the right shoulder, and not so plainly in left upper arm as in right. The breathing is very superficial, chest expansion very slight. Abdominal respiration better than intercostal, but apparently impaired. Patient apparently unable to cough, sneeze or blow the nose. Absolutely unable to roll over in bed. Cannot expel feces. The movements of the right arm fairly good. In left arm, loss of biceps, triceps and deltoid. Movements very feeble in pectoralis major, supinator longus, extensors and flexors of the wrist, interossei, thenar and hypothenar muscles. Movements of neck and face unaffected. All reflexes, deep and superficial, lost. Movements of eyes normal, pupils unaffected.

Twenty-ninth day, breathing better. Facial expression better. Wasting of muscles has appeared, being especially noticeable in the thenar and hypothenar muscles. Pulse has varied between 100 and 140.

Thirty-second day, moves left arm a little more. Has not moved legs since entrance.

Forty-sixth day, can now take a long breath. Respiration still largely abdominal. Voice stronger. Pulse 120, respiration 22. No loss of tactile or temperature sense. Finger movements very good in both hands, and patient extends the right wrist well, the left rather feebly. Flexes the wrist well on the right, rather feebly on the left. Ability to cough slightly improved. No return of reflexes. No power in deltoids or triceps of either side. Complete paralysis of lower extremities persists, with marked wasting and coolness.

I have recently seen the case through the kindness of Dr. Monks, under whose care he is at present, at the City Hospital. It is now nearly three months since the onset of paralysis. The reflexes are still abolished, and no power has returned in the legs. Movement of the left arm has improved somewhat, and the muscles of the hand have begun to fill out. Power of retaining and expelling the feces has improved. There has been slight return of power of erection. Temperature has remained normal; the pulse has varied from 64 to 120; the respiration is constant at 25. The patient reports that he sleeps better and feels stronger. Moderate tenderness and pain persist, especially when he is moved.

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